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Abstract

In order to investigate the association between early life conditions and reproductive and sexuality-related life history outcomes among men, we conducted a meta-analysis that compiled the results of 198 articles. A total of 331 effect sizes drawn from 573 samples were included. The meta-analysis revealed that low family socioeconomic status was associated with early sexual debut ($r = .07$), early first birth ($r = .14$), and early marriage ($r = .03$). There was no significant association between family socioeconomic status and pubertal timing or number of sexual partners. Parental absence was associated with early sexual debut ($r = -.12$), greater number of sexual partners ($r = -.19$), early first birth ($r = -.14$), and early marriage ($r = -.13$). There was no significant association between parental absence and pubertal timing. Small body size before puberty was associated with delayed pubertal timing ($r = -.10$). There was no significant association between adult body size and number of offspring, and between body size at birth and pubertal timing. Small adult body size, greater number of siblings, and older parents were associated with non-heterosexual orientation (r s = .12, .03, and .03 respectively). Factors such as sampling procedure, data collection method, and age cut-off used to measure family structure change influenced the association between some predictors (e.g., family socioeconomic status) and outcomes (e.g., first birth). The findings are discussed in relation to the utility of life history theory for understanding human male reproductive and sexuality-related outcomes.

Keywords: early life conditions; life history theory; life history strategy; meta-analysis; sexual orientation; men

1. Introduction

The life history perspective addresses the problem of how organisms allocate resources and energy to various fitness-enhancing activities including growth, physiological maintenance, and reproduction (Bogin, Silva, & Rios, 2007; Del Giudice, Gangestad, & Kaplan, 2015; Ellis, Figueredo, Brumbach, & Schlomer, 2009). However, as resources spent on one task (e.g., producing offspring) cannot simultaneously be spent on another task (e.g., tissue repair) (Roff, 2002; Stearns, 1992), all organisms face trade-offs regarding how to allocate their resources and energy along their life course (Del Giudice et al., 2015; Ellis et al., 2009). Trade-offs include those between current and future reproduction (Del Giudice et al., 2015; Ellis et al., 2009). For example, organisms which focus on future reproduction allocate more resources to current growth and may develop larger body size, increase skills in intrasexual competition, and enhance the survival of their offspring (Charnov, 1993; Hill & Kaplan, 1999). However, the costs may include low numbers of offspring or higher probability of mortality prior to reproduction (Hill & Kaplan, 1999). Conversely, if organisms allocate resources to earlier reproduction, they may have longer reproductive life spans and more offspring but fewer resources for growth and maintenance (Allal, Sear, Prentice, & Mace, 2004; Ellis, 2004; Furstenberg Jr, Brooks-Gunn, & Chase-Lansdale, 1989; Helle, 2008; Sear, Allal, & Mace, 2004). These “decisions” are made throughout the lifespan and are reflected in an individual's behaviors (including reproductive and sexual behaviors).

In humans, selection has produced a species-typical life history characterized by a long pregnancy, lengthy childhood, high levels of parental investment, low fecundity, and a heterosexual (opposite-sex attracted) sexual orientation pattern (Bogin, 1997; Kaplan, Hill, Lancaster, & Hurtado, 2000; Martin, 2003). However, there appears to be demonstrable individual variation in the speed (fast or slow) of life history strategies and these differences may be influenced by genetic and environmental factors (Ellis, 2004). Growing evidence

suggests that reproductive scheduling among humans is influenced by the environmental conditions experienced during early life (Del Giudice et al., 2015; Ellis et al., 2009; Lummaa & Clutton-Brock, 2002). For example, many studies have reported associations between low birth weight, or low parental investment during the early years, and accelerated pubertal maturation among human females (Quinlan, 2003; Yermachenko & Dvornyk, 2014). Differences in early life experiences may influence subsequent life history strategies (e.g., when to start sexual activity) by constraining an individual's growth or by acting as cues to current or future ecological conditions (Nettle, Frankenhuys, & Rickard, 2013). Thus responding to early life conditions with an accelerated (fast), optimal, or decelerated (slow) life history strategy may be a conditional physiological and behavioral adjustment that has adaptive value (Ellis et al., 2009; Lummaa & Clutton-Brock, 2002).

1.1. Reproductive and sexuality-related life history traits

The application of this framework to human reproduction has focused on the role of early environments (e.g., characterized by harshness, unpredictability, or resource scarcity such as early childhood adversity or unstable family environments) in calibrating reproduction and sexual behavior traits across an individual's lifespan (Del Giudice et al., 2015; Ellis et al., 2009; Winterhalder & Leslie, 2002). For example, a recent meta-analysis has revealed that father absence was associated with girl's earlier menarche (Webster, Graber, Gesselman, Crosier, & Schember, 2014). Another review found that birth weight, environmental hazards (e.g., exposure to endocrine disrupting chemicals), father absence, and low parental socioeconomic status were associated with earlier menarche among girls (Yermachenko & Dvornyk, 2014). Age of menarche, age at first pregnancy, or age at having first child are the best investigated life history traits thus far (Ellis, McFadyen-Ketchum, Dodge, Pettit, & Bates, 1999; Nettle, Coall, & Dickins, 2011; Parent et al., 2003).

What constitutes a “fast” or “slow” life history strategy is debated among life history scholars but the indicators may include pubertal onset, age of sexual debut, age at having first child, age of first marriage, number of sexual partners, and numbers of children (Belsky, 2012; Del Giudice et al., 2015; Ellis et al., 2009). The fastness or slowness is determined simply by the age at which these behaviors begin (e.g., earlier age of menarche is thought to be an indicator of a “fast” life history strategy in which greater resources are allocated to mating effort via the ability to begin reproduction at an earlier age) or number (e.g., more sexual partners or more children could be indicators of a “fast” life history strategy) (Del Giudice et al., 2015; Ellis et al., 2009). We contend that sexual orientation (whether one is heterosexual or non-heterosexual) may also be an indicator of a life history strategy as it is obviously tied to fitness (homosexuality tends strongly to reduce reproductive success but there may be variation in fitness along the spectrum of same-sex contacts; see section below; Bell & Weinberg, 1978).

Several models have been proposed to explain the mechanism underlying the association between adverse early environments and later reproductive life history outcomes. The external predictive adaptive response model suggests that adverse early environments serve as a forecast about the eventual adult environment in which individuals will sexually mature and reproduce (Bateson et al., 2004). The internal predictive adaptive response model suggests that adverse early environments would have negative effect on the individual's future somatic state (Wells, 2011). Thus in both cases, it would be adaptive for the individual to adjust their reproductive schedule for the anticipated environment or future somatic state (Nettle et al., 2013). The energetics theory argues that individuals' physiological mechanisms would track variation in resource availability and adjust physical growth to match on a more event-related basis (Ellis, 2004). Consequently, adverse early environments cause individuals to reserve energy for growth and maintenance and delay reproduction until predictably better

times (Ellis, 2004). Finally, the stress-suppression model argues for a more proximate mechanism of action. This predicts that adverse early environments activate stress responses which suppress the functioning of physiological feedback systems (e.g., the hypothalamic–pituitary–gonadal axis) and depress later reproductive mechanisms (e.g., delaying pubertal timing) (Ellis, 2004). Currently, it is unclear how well the extant empirical evidence in females separate between these competing models. This is because the models overlap, essentially being different versions of the principle of reserving energy and resources for a future reproductive need. The role of parental investment early in life as a critical cue to the future appears critical across all models (Belsky, Steinberg, & Draper, 1991; Ellis, 2004).

The extant research has thus far focused on female reproductive life history traits (for reviews, see Coall, Tickner, McAllister, & Sheppard, 2016; Ellis, 2004; Miller, Benson, & Galbraith, 2001; Webster et al., 2014; Yermachenko & Dvornyk, 2014). This has been influenced by rationales from evolutionary biology including paternity (unlike maternity) uncertainty, that female mammals are the reproductively limiting sex, and that female reproductive physiology is more sensitive to environmental conditions. Yet it is males who show greater variability in reproductive fitness according to Bateman's principles (Arnold, 1994). While environmental conditions alone cannot explain this variability in sex roles, many scholars across the evolutionary sciences agree that incorporating environmental conditions when interpreting animal sex roles and mating systems is important (Arnold, 1994; Janicke, Häderer, Lajeunesse, & Anthes, 2016). There are fewer studies on the role of early life conditions on such life history traits in men and the results of such studies are inconsistent (Bogaert, 2005; James, Ellis, Schlomer, & Garber, 2012). Here we briefly review this literature.

1.2. Family structure changes

Family structure changes (e.g., father absence, mother absence, or both parents absent) are hypothesized to be associated with “fast” life history strategies among men (e.g., early pubertal timing, early sexual debut, early first birth, and early marriage) (Bogaert, 2005; James et al., 2012). However, results are inconsistent. Some studies have found that family structure changes were significantly associated with early pubertal timing (Bogaert, 2005; Kim & Smith, 1998), early sexual debut (Mendle et al., 2009; Pedersen, Samuelsen, & Wichstrøm, 2003), or early first birth (Carlson, VanOrman, & Pilkauskas, 2013; Jaffee, Caspi, Moffitt, Taylor, & Dickson, 2001) among men. Others have revealed the opposite results (Ramirez-Valles, Zimmerman, & Juarez, 2002; Sheppard, Garcia, & Sear, 2015; Sheppard & Sear, 2012), or simply no significant associations between family structure changes and early pubertal timing (Arim, Tramonte, Shapka, Dahinten, & Willms, 2011; James et al., 2012), early sexual debut (Ku, Sonenstein, & Pleck, 1993; Marston, Beguy, Kabiru, & Cleland, 2013), or early first birth (Hanson, Morrison, & Ginsburg, 1989; Winking, Gurven, & Kaplan, 2011).

Prior studies have also used different age cut-offs to determine when men have experienced family structure changes. These range from between 0 and 5 years of age (Miller et al., 1997), 6 and 8 years of age (Sheppard, Schaffnit, Garcia, & Sear, 2014), and 0 and 15 years (Gipson, Hicks, & Gultiano, 2014; Winking et al., 2011). Other studies have used specific ages rather than age-ranges when family structure changes were reported to have been experienced (Michael & Tuma, 1985; Paul, Fitzjohn, Herbison, & Dickson, 2000). In general, it appears that across studies it is the first five to seven years of life that may constitute a sensitive period for the influence of family structure change (and potentially other post-birth factors) on life history (Belsky et al., 2007; Simpson, Griskevicius, Kuo, Sung, & Collins, 2012). This may be due to presence of pre-pubertal hormonal changes including adrenarche, the 5-to-7 years “psychological shift” (e.g., improvements in cognitive

function, language, learning, and social skills), the responsivity of developing neural systems to adverse environmental influences, and increased self-sufficiency during this time (Doom, Vanzomeren-Dohm, & Simpson, 2016; Ellis, 2004). Family structure changes measured at later age period may have less predictive power. Different age cut-offs may be one source of inconsistency between studies and this requires further investigation.

1.3. Family socioeconomic status (SES)

While prior research shows that low parental or family SES is associated with increased mortality or morbidity and resource scarcity (Belsky, Schlomer, & Ellis, 2012; Griskevicius, Delton, Robertson, & Tybur, 2011), the results for reproductive life history strategies among men is inconsistent. Some studies have found that low SES was significantly associated with early pubertal timing (Chasiotis, Scheffer, Restemeier, & Keller, 1998), early sexual debut (Paul et al., 2000; Valle, Røysamb, Sundby, & Klepp, 2009), or early first birth (Fagot, Pears, Capaldi, Crosby, & Leve, 1998; Hanson et al., 1989) among men. Others have revealed the opposite results (Gipson et al., 2014; Sheppard et al., 2015), or no significant associations with early pubertal timing (James et al., 2012; Sheppard & Sear, 2012), early sexual debut (Davis & Friel, 2001; Smith, 1997), or early first birth (Waynforth, Hurtado, & Hill, 1998; Xie, Cairns, & Cairns, 2001).

Studies also differ in the manner in which they measure family SES. Some studies have used a single indicator such as mother's or father's education (Anderson, 2015; Valle et al., 2009), mother's or father's occupation (Michael & Tuma, 1985; Wellings et al., 2001), or family income (Davis & Friel, 2001; Fagot et al., 1998). Others have used a combination of these as a proxy for family SES (Chasiotis et al., 1998; James et al., 2012). It is possible that some aspects of family SES (e.g., family income) are better indicators of early environmental harshness or resource scarcity than others. Thus, different family SES measurements may contribute to the inconsistent results.

1.4. Body size

Body size (including birth weight, birth height as well as post-birth factors such as adult size) are potential indicators of adverse early environmental conditions and low physiological parental investment, although also strongly associated with nutritional and SES status (Blumenshine, Egerter, Barclay, Cubbin, & Braveman, 2010; Drewnowski & Specter, 2004; Krieger et al., 2003; Paeratakul, Lovejoy, Ryan, & Bray, 2002). Males with small body size are predicted to show faster reproductive life histories. However, results of studies are inconsistent. Some show that small body size before puberty was significantly associated with early pubertal timing (Vizmanos & Marti-Henneberg, 2000), and that small adult body size was significantly associated with greater numbers of offspring (Kirchengast & Winkler, 1995). Others have revealed the opposite results (Campbell, Gillett-Netting, & Meloy, 2004; Pawlowski, Dunbar, & Lipowicz, 2000), or no significant associations with pubertal timing (Sheppard & Sear, 2012), and number of offspring (Sear, 2006).

1.5. Other early life factors

Prior studies have also investigated the effects of other early life factors. For example, studies have suggested that parents may bias their resource allocation to their children in order to optimize the reproductive success of some of their children because of limited resources. Thus, the competition of resources between siblings may be more intense with increasing number of siblings (Faurie, Russell, Lummaa, & Sear, 2009; Rickard, Russell, & Lummaa, 2007). Studies have also suggested that having teenage parents or older parents was associated with adverse birth outcomes (e.g., preterm birth and low birth weight) (Sartorius & Nieschlag, 2009), which may increase the later mortality or morbidity of offspring. Studies have also found that preterm birth children were at higher risk for mortality or morbidity (McMillen & Robinson, 2005). Thus, men exposed to conditions of either preterm birth, having teenage or older parents, or greater number of siblings were hypothesized to be

associated with “fast” reproductive outcomes. Prior studies exploring associations between these factors and later reproductive life history strategies among men have yielded inconsistent results (See Supplemental Table 1).

1.6. Sexual orientation and life history

Sexual orientation refers to the degree to which a person is attracted to same- or opposite-sex members (Bailey et al., 2016). Early life conditions may be associated with variation in male sexual orientation. For example, number of siblings (particular number of older brothers) and low birth weight are associated with male homosexuality (Blanchard, 2012; Bogaert & Skorska, 2011). While sexual orientation is generally not considered by life history researchers (perhaps because exclusive homosexuality is non-reproductive in Western societies), it is of intrinsic scientific interest to ask whether early life conditions associated with other reproductive outcomes (e.g., number of offspring) are similar for sexual orientation. We might also hypothesize that heterosexuality is an optimal life history strategy given the species-typical pattern of sexual interests in humans is towards the opposite sex. Non-heterosexual orientation (e.g., homosexual/bisexual) may represent a “fast” life history or be a by-product of other life history traits.

Sexual orientation is often measured using one or more of four components, including sexual behavior, sexual identity labels, sexual attraction, or physiological measures (e.g., genital arousal) (Bailey et al., 2016). The consistency among sexual orientation components is modest, perhaps because self-reported sexual identity and sexual behavior components are far more environmentally malleable than sexual attractions and physiological responses (Bailey et al., 2016; Savin-Williams, 2009). For example, actions of people who engaged in same-sex behavior may reflect various motivations other than sexual attraction (Bailey et al., 2016). Thus, the prevalence of non-heterosexuality depends somewhat on the component used to measure sexual orientation and the criteria used to classify individuals as non-

heterosexual (Bailey et al., 2016). The observation that individuals with infrequent homosexual behavior (actual sexual contacts) and attractions are more common than those with substantial or exclusive (persistent and strong) homosexuality may permit some direct reproduction to occur because heterosexual contacts still dominate in the former group (Bailey et al., 2016). For example, the percentage of adults reporting a history of any same-sex sexual contacts ranged from 6.9% to 8.8%. This exceeds those reporting a non-heterosexual identity (or social label) by ratios ranging from 2:3 to 3:3 (Gates, 2011). From an evolutionary perspective, any reproductive contact resulting in offspring is better than none (However small given homosexuality's association with low direct reproduction). Thus, here we operationalize sexual orientation as 'heterosexual' versus 'non-heterosexual', to encompass the fact that the latter group may comprise potentially reproductive and non-reproductive sexual contacts.

Studying sexual orientation from a life history perspective may also enhance our understanding of its origins. For example, the dominant theoretical explanation for male sexual orientation is hormonal. This proposes that lower pre-birth androgen exposure may be associated with homosexuality in men (Ellis & Ames, 1987; Rahman, 2005). While there is some support for prenatal androgen theory it does not explain why homosexual men should experience varying levels of exposure in the first place (Bailey et al., 2016). Similar, the finding that number of older brothers is strongly associated with male [but not female] homosexuality is theorized to be due a pre-birth maternal immune response (Blanchard, 2004). But no attempt is made to understand what triggers that response. Twin studies show that a substantial number of identical twins do not share the same sexual orientation (yet share the same genotype) and thus non-shared environmental factors (including the prenatal environment) must play a role (Bailey, Dunne, & Martin, 2000). We propose that early life conditions suggested by life history theory may constitute one source of these non-shared

factors in the development of sexual orientation. Differences in early life conditions (pre-birth or post-birth) may, for example, contribute to hormonal processes or maternal immunity and then bias subsequent sex differentiation of the developing brain in utero affecting circuitry related to sexual orientation (Rahman, 2005).

1.7. The current study

The objective of this study was to synthesize existing literature and quantify the association between early life conditions (family SES, family structure change, body size, parental age, number of siblings, and other minor factors including household moves) and reproductive and sexual behavior-related life history in men. The current study is, to our knowledge, the first meta-analysis of this body of work in men. Previous studies were identified by a systematic search and effect sizes were computed using meta-analysis. Supplemental Table 1 presents the number of studies that have demonstrated significant and non-significant results among male samples separately by early life factors and reproductive life history traits. The details of studies included in Supplemental Table 1 are listed in Supplemental Table 2. Different studies that used the same data were counted once in Supplemental Table 1. Prior research has suggested that meta-analyses based on a few papers have lower statistical power (Valentine, Pigott, & Rothstein, 2010). In order to increase the statistical power of this study, the current meta-analysis only included associations between early life conditions and life history where there were seven or more independent studies.

2. Method

2.1. Selection of studies

We used two search methods to identify eligible articles published between January 1970 and October 2016. Firstly, we searched the electronic databases PsychInfo, PubMed, and ProQuest, for articles examining the influences of family structure changes, family SES, body size, parental age, and number of siblings on pubertal timing, sexual debut, first birth, early

marriage, number of sexual partners, number of offspring, and sexual orientation among men, using the terms listed in Supplemental Text 1. Secondly, we examined the reference lists of the collected articles.

To be included in this meta-analysis, articles had to meet the following inclusion criteria: (a) male samples; (b) their main or secondary objective was to investigate the influences of family structure changes, family SES, body size, parental age, and number of siblings on pubertal timing, sexual debut, first birth, early marriage, number of sexual partners, number of offspring, and sexual orientation; (c) they provided sufficient data, including correlation coefficients, regression coefficients (*beta*), odds ratios, hazard ratio, risk ratio, or other statistics, to determine the effect size; (d) the data that the articles provided were not repetitive.

2.2. Coding of studies

(a) Family SES (family income, mother's education, father's education, mother's occupation, father's occupation, combination of two or more of these aspects); (b) family structure (father absence, mother absence, either or both parents absence); (c) the age cut-off used to measure family structure changes (0-8, 8-18, 0-18); (d) body size (height, weight, BMI); (e) Number of siblings (number older brother, number of older sister, number of younger brother, number of younger sister); (f) parental age (maternal age or paternal age); (g) pubertal timing measurement (one indicator e.g., voice change, pubic hair, or spermatarche, or combination of two or more of these indicators); (h) offspring type (alive, ever born, unknown); (i) the country in which the study was conducted; (j) sample size; (k) the sampling procedure (probabilistic or non-probabilistic); (l) data collection method (retrospective or longitudinal study).

2.3. Meta-analytic procedures

The meta-analysis was performed using the Comprehensive Meta-Analysis 3.0. First, we used the correlation coefficient (r) as a measure of effect size. In order to produce standardized effect sizes and avoid potential bias in the estimate of effect sizes, we used the Fisher's procedure for transforming r to Z scores to allow pooling (After pooling, the Z scores were back transformed to r to facilitate interpretation of the results). Second, we used the box plots to identify the outliers. Third, we computed the combined effect sizes using the random effects model, and tested the heterogeneity of the studies by means of the I^2 statistics. Analyses were performed both including and excluding studies where the effect sizes were judged to be outliers. Fourth, in order to explore whether the heterogeneity can be explained by some of the methodological variations between studies, we conducted moderator analyses by means of the Q statistics using the random effects model¹. Fifth, we performed sensitivity analyses to identify potential publication bias.

The studies included in the current meta-analysis provided diverse effect size indicators. For studies that provided *beta*, we used the formula proposed by (Peterson & Brown, 2005) to transform *beta* to r . The formula is $r = 0.98\beta + 0.05\lambda$, where λ is a variable that equals 0 when β is negative and 1 when β is non-negative (Peterson & Brown, 2005). For studies that provided odds ratio, we used the tetrachoric approximation proposed by (Digby, 1983) to transform odds ratio to r . The formula is $r = (\text{odds ratio}^{3/4} - 1) / (\text{odds ratio}^{3/4} + 1)$ (Bonett, 2007). For studies that provided values of t , Cohen's d , F , and χ^2 , we used the formulas proposed by (Rosenthal & DiMatteo, 2001) to transform these to r . The formulas are $r = (t^2 / (t^2 + df))^{1/2}$, $r = (d^2 / (d^2 + 4))^{1/2}$, $r = (F / (F + df_{error}))^{1/2}$, and $r = (\chi^2 / N)^{1/2}$, respectively (Rosenthal & DiMatteo, 2001). In the current study, we treat hazard ratio equals to risk ratio. There is no standard approach to transform risk ratio directly to r . Thus, for studies that provided risk or hazard ratio and enough information to construct the 2×2 contingency table, we used the formula proposed by (Bonett, 2007) to transform risk or hazard ratio to odds

ratio, then used the tetrachoric approximation proposed by (Digby, 1983) to transform odds ratio to r . The formula is odds ratio = risk ratio * $(1 - p_o) / (1 - \text{risk ratio} * p_o)$ where p_o is the incidence of the outcome in the reference group (Bonnett, 2007). For studies that provided risk or hazard ratio and 95% confidence interval (CI) but did not provide enough information to construct the 2×2 contingency table, we used the formula proposed by (Altman & Bland, 2011) to transform risk or hazard ratio to Z scores, then we used the formula proposed by (Rosenthal & DiMatteo, 2001) to transform Z score to r (Roberts, Kuncel, Shiner, Caspi, & Goldberg, 2007). For studies that provided risk or hazard ratio and SE but did not provide enough information to construct the 2×2 contingency table, we used the formula proposed by (Hackshaw, 2009) to compute the 95% CI, then we used the formula described above to transform it to r (Altman & Bland, 2011; Roberts et al., 2007; Rosenthal & DiMatteo, 2001). For studies that provided risk or hazard ratio but did not provide enough information to construct the 2×2 contingency table, 95% CI, or SE , we used $r_{\text{equivalent}}$ proposed by (Rosenthal & Rubin, 2003), which is computed either from exact p values and sample size or conservative p values and sample size.

Studies often presented bivariate relations of predictor and life history strategies, as well as multivariable analyses including all independent variables and controlling for potential covariates. The results of multivariable analyses were coded for the current meta-analysis. In addition, where studies divided family SES into more than two groups (e.g., college degree, high school, elementary school, and no qualification), the effect sizes for the lowest SES were coded for the current meta-analysis.

When studies used two or more different measures of an outcome (e.g. different indicators of pubertal timing), the averaged effect size was computed. Research has suggested that the consistency among sexual orientation components is modest, perhaps because self-reported sexual identity and sexual behavior components are far more environmentally malleable than

sexual attraction (Bailey et al., 2016). Thus, we chose to code the results for sexual attraction where this was reported, otherwise sexual identity or sexual behavior were used if these were the only components available.

When studies provided multiple indicators of a particular class of early life conditions (e.g., family income, mother's education, and father's education as indicators of family SES), we used the approach of Borenstein, Hedges, Higgins, & Rothstein (2009) to combine the effect sizes and variances across indicators within each study. This avoids double counting of data from the same individuals when calculating the pooled estimate. For the combined variance, intercorrelations among the indicators within a class are required but typically not reported by studies. Therefore, intercorrelations between indicators used in calculating the combined variance were derived from two large longitudinal datasets (ALSPAC and Understanding Society)². The intercorrelations are shown in Supplemental Table 3. On some occasions studies had included multiple indicators of an early life condition in a linear regression model and reported the R^2 for the model. Where this was the case, we computed the multiple correlation coefficient (e.g., $\sqrt{R^2}$) as the combined effect size, since applying the Borenstein method to the standardised beta's is known to underestimate the true effect .

Effect sizes in the current meta-analysis were reported as rs^3 . For family structure change, a positive r represents family structure change was associated with "slow" life history strategies, while a negative r represents family structure change was associated "fast" life history strategies. For family SES, body size, parental age, and number of siblings, a positive r represents low family SES, small body size or obesity, older parents, or greater number of siblings was associated with "fast" life history strategies, while a negative r represents low family SES, small body size or obesity, older parents, or greater number of siblings was associated "slow" life history strategies. However, some prior studies used different groups (e.g., father absence or father presence) as the reference, and the direction of

the r s produced by the formulas presented above were not always consistent. Thus, we changed the direction of the transformed r for some studies (see Supplemental Table 4).

3. Results

3.1. Characteristics of studies

According to the inclusion criteria, the final sample of the current meta-analysis comprised 198 articles across 39 countries. A total of 331 effect sizes and 960,146 participants were included (see Supplemental Table 5).

3.2. Association between early life factors, and reproductive and sexual life history outcomes

Outliers were detected using the box plots (see Supplemental Text 2). Sensitivity analyses revealed no substantive differences either in terms of the pooled effect sizes (r s) or heterogeneity test between analyses including and excluding outliers. Thus results presented below are reported including outliers.

Table 1 represents the pooled effect sizes separately by early life conditions and life history outcome variables. Family structure change was the strongest predictor of life history outcome variables. Parental absence was found to be associated with early sexual debut, early first birth, early marriage, and greater numbers of offspring, with r ranging from $-.19$ to $-.12$, all $ps < .01$. Family SES was also a strong predictor. Low family SES was found to be associated with early sexual debut, early first birth, and early marriage, with r ranging from $.03$ to $.14$, all $ps < .05$. For sexual orientation, small adult body size, greater number of siblings, and having older parents was associated with non-heterosexual orientation, with r ranging from $.03$ to $.12$, all $ps < .001$.

Except samples investigating parental age and sexual orientation ($I^2 = 0.00$), the heterogeneity in the samples was very high (the value of I^2 ranging from 78.72% to 99.27%), indicating that over 79% of residual variation was attributable to statistical heterogeneity in the effect sizes between samples. The impact of design heterogeneity between studies on

statistical heterogeneity in effect sizes was assessed by examining potential moderators of the effect size between studies.

3.3. Analysis of moderators

Table 2 to Table 4 represents the results of moderator analyses separately for family structure change, body size for gestational age, and number of siblings. Supplemental Table 6 to Supplemental Table 8 represents the results of moderator analyses separately for family SES, childhood or adult body size, and parental age.

For the association between family structure change and first birth, the estimated r differed according to the age cut-off used to measure family structure change, $Q(2) = 9.74, p < .01$. The lowest r was found in samples measuring family structure change under 8 years of age, whereas the highest was found in samples measuring family structure change from birth to 18 years old.

For the association between sexual orientation and number of siblings, the estimated r differed according to the sibling type, $Q(3) = 27.03, p < .001$. The highest r was found in samples measuring number of older brothers (consistent with prior literature), whereas the lowest was found in samples measuring number of younger sisters.

Retrospective studies were more likely to reveal an association between low family SES and early first birth, and parental absence and early marriage compared to longitudinal studies (all $ps < .001$). Studies using non-probabilistic samples were more likely to report an association between low family SES and early first birth compared to studies using non-probabilistic samples ($p < .001$).

3.4. Publication bias

Begg and Mazumdar's rank correlation test and Egger's linear regression test were conducted separately by predictors and outcomes to detect the publication bias. Except studies examining the association between adult body size and sexual orientation, the results

of Begg and Mazumdar's rank correlation test (the value of p ranging from .267 to .956) and Egger's linear regression test (the value of p ranging from .174 to .994) suggested this meta-analysis lacked publication bias.

4. Discussion

The current meta-analysis produced five main findings. Firstly among men, low family SES was associated with early sexual debut, early first birth, and early marriage, whereas there were no significant associations between family SES and pubertal timing or number of sexual partners. Secondly, parental absence was associated with early sexual debut, greater number of sexual partners, early first birth, and early marriage. Thirdly, small body size before puberty was associated with delayed pubertal timing, but there was no significant association between body size for gestational age and pubertal timing, [and between adult body size and number of offspring](#). Fourthly, small adult body size, greater number of siblings, and having older parents was associated with non-heterosexual orientation. However, effect sizes were uniformly small in magnitude across the associations. Finally, the sampling procedure, data collection method, and age cut-off used to measure family structure change influenced the association between some predictors (e.g., family SES) and outcomes (e.g., first birth). Better quality studies (e.g., longitudinal) tended to report weaker associations than poorer quality studies (e.g., retrospective).

The findings are consistent with growing evidence that early life conditions (including family SES [and](#) parental absence) influence reproductive scheduling (e.g., age at menarche and age of first pregnancy) among women (Webster et al., 2014; Yermachenko & Dvornyk, 2014). Our results provide some support for the prediction from life history theory that adverse early life or environmental conditions are associated with an accelerated life history strategy in men (Bogaert, 2005; Del Giudice et al., 2015; James et al., 2012). This may include allocating resources and making “decisions” to focus on current reproduction and the

quantity of the offspring to maximize fitness under harsh, unpredictable, or resource scarce conditions (Del Giudice et al., 2015; Ellis et al., 2009). Post-birth and pre-birth factors in common to both sexes in predicting reproductive and sexual behavior outcomes (based on this analysis and prior work on women) appear to include family SES, parental absence, and body size. The most robust effect sizes found here appear to be for the association between parental absence and number of sexual partners and early first birth, and between low family SES and early first birth. The association between early life factors and pubertal timing among men was inconsistent and thus not in line with previous findings among women (e.g., where father absence is robustly associated with early menarche; Webster et al., 2014). For example, we found that small body size before puberty were associated with delayed pubertal timing, and there was no significant association between family SES, [parental absence](#), body size for gestational age, and pubertal timing. This is inconsistent with predictions from life history theory frameworks.

In relation to sexual orientation, we found that small adult body size, greater numbers of siblings, and having older parents was associated with non-heterosexuality. However, the number of studies for parental age was small. In contrast, the findings for number of siblings are consistent with a large body of work for a fraternal birth order effect in relation to male sexual orientation (Blanchard, 2004). That is, number of older brothers most likely derives our finding regarding number siblings. Here, we have proposed that non-heterosexuality (a broader definition of same-sex sexuality that incorporate heterosexual or potentially reproductive sexual contacts) may represent a “fast” life history strategy or a by-product associated with other life history traits due to exposure to common early life conditions. The observation that body size in particular was associated with sexual orientation and pubertal timing in our analysis suggest that there are early life conditions associated with a range of fast reproductive outcomes. The link between early life conditions, sexual orientation, and

pubertal timing may involve some “third” factors such as prenatal androgen exposure or maternal immunity responses (Rahman, 2005).

The present findings may have theoretical implications for life history theory frameworks. Firstly, the type of reproductive or sexual behavior outcome (e.g., pubertal timing, sexual debut, first birth, number of sexual partners, and sexual orientation) appears critical. Thus, life history models should attend to the issue of specificity. Some models view early adversity as detrimental to development irrespective of context or type of outcome, or predict generic fitness disadvantages that are less amenable to modification throughout the life-course (Grafen, 1988; Jones, 2005). The current results suggest that the experience of early life conditions may operate in a more facultative manner given the different patterns of associations for different reproductive outcomes (Nettle et al., 2013; Wells, 2012). The finding that age moderated the association between some predictors (e.g., [family structure change](#)) and reproductive outcomes (e.g., first birth) support the notion of this plasticity in response to early and later developmental periods.

The small, if robust, associations found here are consistent with the possibility that other causal factors influence both our independent and dependent variables, such as genetics (e.g., Barbaro, Boutwell, Barnes, & Shackelford, 2017). Specifically, individual differences in reproductive and sexual behavior as well as early life conditions may be more strongly influenced by genetic compared to environmental variation, and there may be genetic correlations between these factors. Twin studies show moderate genetic influences on several reproductive and sexual behavior traits, including pubertal onset, sexual debut, and sexual orientation; although the estimates differ depending on the outcome highlighting the importance of specificity (Bailey et al., 2016; Dick, Rose, Pulkkinen, & Kaprio, 2001; D’Onofrio et al., 2006; Ellis, Schlomer, Tilley, & Butler, 2012; Mustanski, Viken, Kaprio, Pulkkinen, & Rose, 2004; Silventoinen, Haukka, Dunkel, Tynelius, & Rasmussen, 2008). A

recent study has found that even a small genetic correlation between father absence and age at menarche in women could confound the phenotypic correlation between these two variables (Barbaro et al., 2017).

In addition, a large body of behavior genetic research shows small or negligible influences of the shared environment (which includes the family environment) and a stronger role for non-shared or unique environmental factors (factors outside the family environment including peer socialization and other biological influences) (Polderman et al., 2005). However, our findings are largely silent on whether genetic, shared or non-shared environmental factors are most important. In terms of ostensibly shared environmental influences, family structure changes were more strongly associated (in terms of effect sizes) with sexual debut, early marriage, and number of sexual partners than was family SES. Both family structure changes and family SES were strongly associated with first birth. Neither family structure change nor family SES were as strongly associated with pubertal timing as they were with the other outcomes. Body size (a factor likely influenced by genetic and non-shared environmental factors) was more strongly associated with sexual orientation than the other outcomes. Thus, the influence of genetic, shared environment and non-shared environmental factors is by no means equivalent across our associations. Again, the type of reproductive and sexual dependent variable appears critical. Although the degree of environmental and genetic influence on life history activities is complex, we encourage future researchers to explore genetic confounds that might be responsible for any association with early life conditions and reproductive outcomes among men (Zietsch, 2016).

Other explanations for our results should also be considered, such as peer socialization and developmental psychopathology (e.g., conduct disorder). It is possible that children and adolescents with absent fathers receive less parental monitoring and supervision, which may increase their affiliation with deviant peers who engage in early or risky sexual activities

(DelPriore, Schlomer, & Ellis, 2017). Studies also suggest that adolescents who experienced adverse early environments are more likely to engage in social deviant behaviours and have conduct problems, some of which are associated with early sexual debut and greater numbers of sexual partners (Brumbach, Figueredo, & Ellis, 2009; Wenner, Bianchi, Figueredo, Rushton, & Jacobs, 2013). Whether these associations are due to genetic or environmental correlations is unknown. However, it would be useful for future life history studies to explore the role of peer socialization or the presence of conduct problems or other developmental psychopathology as mediators or moderators.

Variation in participant factors may have also influenced the estimates reported here. These include social desirability biases and cultural sensitivities between the study samples that may influence reporting of reproductive and sexual behaviors. Cultural factors may also influence the strength or “severity” of some of the associations. For example in India women control fewer resources than do men, and so father absence may have strong negative influences of children’s reproductive success because paternal investment is less substitutable (Shenk & Scelza, 2012). Mismatches between early developmental environments and later ones may also be important. For example, a mismatch between early nutritional poverty and later nutritional excess has been found to be associated with a greater risk of health problems associated with somatic growth and metabolism (Gluckman, Hanson, & Beedle, 2007; Godfrey, Gluckman, & Hanson, 2010). Thus variations or shifts from early resource-poor to later resource-rich contexts may modulate developmental trajectories (Gluckman et al., 2009).

We also showed that over 79% of the residual variation was attributable to heterogeneity between samples and that some methodological differences between studies could be attributed to the heterogeneity. In addition, poorer quality studies (e.g., retrospective) reported stronger associations than did better quality studies (e.g., longitudinal). Studies using non-probabilistic samples (which are less representative of the population at large) tended to

find associations between family SES and first birth compared to studies using probabilistic samples. Retrospective studies were more likely to reveal an association between family SES and early first birth, [and between parental absence and early marriage](#) than longitudinal studies. Non-probabilistic samples may be biased by small sample size and lack of representativeness, while the results of retrospective studies are biased by recall error, social desirability biases, and cannot determine causality. In addition, pre-existing longitudinal studies on male life history were restricted to variables included in those studies, and those variables may be less sensitive regarding age and outcomes since they were included a long time ago with different study aims instead of targeting specifically investigating life history questions. This is important as some studies and life history theorists have suggested that the first five or seven years of life are the most sensitive period for the calibrated adjustment of life history strategy in response to early life conditions (Belsky et al., 2007; Simpson et al., 2012). Future studies should endeavor to study this period in development rather than later ages, or test whether late-childhood or adolescent adversity moderates or confounds the impact of early childhood adversity on reproductive outcomes. In general, while our pooled estimate was less stable the range of observed effect sizes (see Table 1) showed clear trends in the predicted directions. This was aided by the relatively large sample sizes in most studies analyzed here and our attempt to examine a broad range of fitness-relevant indicators, including indirect and direct measures (e.g., first birth, early marriage, number of offspring). However, it is important to note that the literature examining associations between early life conditions and number of offspring (which is a direct measure of fitness) was scant. Thus, the present meta-analysis relied mostly on studies in which “fitness” is an indirect measure made up of other reproductive, mating, and sexual behaviors.

The current meta-analysis had several other limitations. First, the studies included in the current meta-analysis provided diverse effect size indicators (e.g., r , risk ratios, and hazard

ratio). We used different formulas to transform these indicators to r . However, some transformations produced relatively low values of r (e.g., the $r_{\text{equivalent}}$ computed using p values and large sample size). Second, the heterogeneity between studies included in the current meta-analysis was high. Methodological variation between studies (e.g., in sampling procedure) could only partially explain the heterogeneity. Third, it may be problematic that we categorized studies measuring family structure change during both earlier and later childhood into 0 - 18 years (e.g., experience father absence between 6 and 12 years old). Fourth, the current meta-analysis focused on modern industrial societies with relatively low fertility, access to modern contraceptive technology, and other lifestyle features (e.g. energy-rich diets that may influence body size and pubertal timing) that were less common over human evolutionary history. Finally, we did not include the effect of childhood gender nonconforming (CGN) behavior on male sexual orientation. CGN is a powerful developmental predictor of adult sexual orientation as found in retrospective and prospective studies (e.g., Bailey & Zucker, 1995; Jones, Robinson, Oginni, Rahman, & Rimes, 2017; Xu & Zheng, 2017). Its place in the context of life history theory framework is somewhat unclear. For example, early life factors could promote CGN, which then cascades into differences in adult sexual orientation (in which case the early life factors are “closer” in the causal sense to CGN rather than to sexual orientation). Alternatively, CGN could act as a behavioral proxy marker for a common underlying proximate mechanism, such as prenatal androgen exposure. Thus, CGN may serve as a mediator or moderator variable. This requires exploration in future work.

Given that methodological variation could affect the association between family SES, family structure change and life history strategy, future studies in this field should use probabilistic and longitudinal samples (e.g., those that directly measure early life conditions before 8 years of age and follow up individuals to adulthood). Our analysis is also silent on

possible third variables or proximate causal factors, which may act as mediators for the associations found. For example, it is possible that some of our measures of early life conditions (e.g., family SES) act as mediators between other conditions (e.g., low body size) and later reproductive outcomes. Socioeconomic status, owing to its close association with resource availability and acquisition, should be studied carefully. Studies that explore proximate factors such as personality, health behaviors, and risk-level are also needed. For example, not everyone who experiences early adversity will develop a fast life history strategy and those who experience high levels of initial adversity may be at increased risk of re-experiencing it during later sensitive periods (Sheppard, Pearce, & Sear, 2016). Again, longitudinal studies are best suited and powered to test for direct and indirect mediation or moderation pathways.

In sum, this meta-analysis offers some support for the central tenet of the life history perspective that early life conditions are associated with fast and slow reproductive and sexual behavior outcomes in humans. However, this conclusion should be treated with caution given the high heterogeneity reported above and the small, if robust, effect sizes. This analysis in men adds to the accumulating evidence found in women. Together they suggest that the mechanisms of conditional adjustment to the demands of local environment or ecology could exert important influences on human reproduction. Exactly how the early-life influences lead to reproductive differences requires further investigation and such future studies should ideally be longitudinal in design (to help isolate causal pathways). In relation to sexual orientation, our analysis here suggests that life history may be a useful framework to understand variation in this sexual phenotype (one that is directly tied to fitness outcomes). We show that some early life conditions associated with sexual orientation may overlap with other reproductive life history traits and thus offer a way to elucidate common developmental proximate mechanisms responsible for these traits.

Note

¹ In the moderator analysis it was not always possible to use effect sizes combined across indicators within the same domain. For example, when testing whether effect sizes differed across indicators within a domain, such as family income versus father's education. Where data for individual indicators were used, we ensured that standard errors were not biased by adjusting the sample size.

² We performed sensitivity analyses to check whether the pooled effect sizes would change when different intercorrelations were used ($r = .00, .50$, and $.99$). Sensitivity analyses showed that the impact on the pooled effect sizes was negligible (See Supplemental Table 9).

³ We encourage readers to focus on the pooled effect sizes and the uncertainty around these effect sizes, as indicated by confidence intervals, rather than statistical significance tests due to the number of tests conducted without correction for multiple testing (Schmidt & Hunter, 2015).

Competing interest.

The authors declare no competing interests.

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